

THAT WHICH IS CLAIMED:

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A1
5 1. A method of enhancing the cytotoxic effects of an antineoplastic chemotherapeutic agent, comprising administering a therapeutically effective amount of NF- κ B inhibitor in conjunction with the administration of the chemotherapeutic agent, whereby the cytotoxic effect of said chemotherapeutic agent is increased compared to that which would occur in the absence of NF- κ B inhibitor.

2. A method according to claim 1 wherein said NF- κ B inhibitor is administered simultaneously with said chemotherapeutic agent.

SUB C3
3. The method of claim 1 where said chemotherapeutic agent is selected from the group consisting of daunorubicin, vincristine, and irinotecan.

5 4. A method according to claim 1, wherein said NF- κ B inhibitor is selected from the group consisting of the super-repressor I κ B α , NF- κ B inhibiting proteasome inhibitors, NF- κ B inhibiting ubiquitin inhibitors, NF- κ B inhibiting proteasome peptidases, NF- κ B inhibiting proteases and antisense oligonucleotides that bind to mRNA encoding NF- κ B.

5 5. A method according to claim 1 wherein said NF- κ B inhibitor is delivered to said cell by transfecting said cell with a vector encoding said NF- κ B inhibitor.

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5 6. A method of enhancing chemotherapeutic cytotoxicity in a subject treated with an antineoplastic chemotherapeutic agent, comprising administering to the subject a therapeutically effective amount of NF- κ B inhibitor in conjunction with the administration of the chemotherapeutic agent, whereby the cytotoxic effect of said chemotherapeutic agent is increased compared to that which would occur in the absence of NF- κ B inhibitor.

7. A method according to claim 6 wherein said NF- κ B inhibitor is administered simultaneously with said chemotherapeutic agent.

8. A method according to claim 6 wherein said chemotherapeutic agent is selected from the group consisting of daunorubicin, vincristine and irinotecan.

9. A method of enhancing the cytotoxic effect of $\text{TNF}\alpha$, comprising administering a therapeutically effective amount of NF- κ B inhibitor in conjunction with the administration of $\text{TNF}\alpha$, whereby the cytotoxic effect of $\text{TNF}\alpha$ is increased compared to that which would occur in the absence of NF- κ B inhibitor.

10. A method according to claim 9 wherein said NF- κ B inhibitor is administered simultaneously with $\text{TNF}\alpha$.

11. A method of enhancing chemotherapeutic cytotoxicity in a subject treated with $\text{TNF}\alpha$, comprising administering to the subject a therapeutically effective amount of NF- κ B inhibitor in conjunction with the administration of $\text{TNF}\alpha$, whereby the cytotoxic effect of $\text{TNF}\alpha$ is increased compared to that which would occur in the absence of NF- κ B inhibitor.

12. A method according to claim 11 wherein said NF- κ B inhibitor is administered simultaneously with $\text{TNF}\alpha$.

13. A method of screening a compound for the ability to reduce the anti-apoptotic protective effects of an NF- κ B induced anti-apoptotic gene, comprising:

exposing a population of test cells to an anticancer treatment and a test compound;

determining cell viability after said exposure;

where reduced cell viability, compared to that which occurs in the absence of said test compound, indicates that said test compound reduces the anti-apoptotic effects of an NF- κ B induced anti-apoptotic gene.

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A3

add C6